

All Databases

PubMed

Nucleotide

Protein

Genome

Structure

OMIM

PMC

Journals

Book

Search PubMed

for

Go

Clear

☒ Limits

Preview/Index

History

Clipboard

Details

2132.106

Examiner

copy

reference 5

Limits: Publication Date from 1995 to 2000

Display Abstract

Show 20

Sort by

Send to

About Entrez

Text Version

All: 1 Review: 0 ☒

Entrez PubMed

Overview

Help | FAQ

Tutorial

New/Noteworthy

E-Utilities

PubMed Services

Journals Database

MeSH Database

Single Citation Matcher

Batch Citation Matcher

Clinical Queries

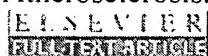
Special Queries

LinkOut

My NCBI (Cubby)

☐ 1: Atherosclerosis. 2000 Sep;152(1):229-37.

Related Articles, Links



Inefficiency of insulin therapy to correct apolipoprotein A-I metabolic abnormalities in non-insulin-dependent diabetes mellitus.

Duvillard L, Pont F, Florentin E, Gambert P, Verges B.

INSERM U 498-Metabolisme des lipoproteines humaines et interactions vasculaires, Faculte de Medecine, 21033, Dijon, France.
laurence.duvillard@u-bourgogne.fr

Related Resources

Order Documents

NLM Catalog

NLM Gateway

TOXNET

Consumer Health

Clinical Alerts

ClinicalTrials.gov

PubMed Central

Non-insulin-dependent diabetes mellitus (NIDDM) is associated with low high density lipoprotein (HDL) cholesterol and apoA-I, related to an increased apoA-I fractional catabolic rate. This stable isotope kinetic experiment, using L-[1-(13C)] leucine, was designed to study the effect of insulin therapy on HDL apoA-I and A-II metabolism in poorly controlled NIDDM patients. A kinetic study was performed in five control subjects and in six NIDDM patients before and two months after the introduction of insulin therapy. ApoA-I and A-II were modelled using a monoexponential function. Insulin treatment was able to correct neither the low HDL apoA-I concentration observed in NIDDM patients (1.14 ± 0.19 vs. 1.16 ± 0.12 g l⁻¹ (-1) (controls: 1.33 ± 0.14)), nor the HDL apoA-I hypercatabolism (0.39 ± 0.11 vs. 0.34 ± 0.05 pool d(-1), (controls: 0.23 ± 0.01 , $P < 0.01$)). HDL apoA-I production rate was increased in NIDDM patients compared to control subjects and was not modified by insulin (0.45 ± 0.12 vs. 0.39 ± 0.08 g d(-1) l(-1), (controls: 0.31 ± 0.04 , $P < 0.05$)). HDL apoA-II kinetic parameters were initially not significantly different between NIDDM patients and control subjects, and were not modified by insulin. The decreased insulin sensitivity, assessed by the insulin suppressive test, was not modified by insulin therapy in NIDDM patients. HDL apoA-I fractional catabolic rate was significantly correlated to HDL triglyceride/cholesterol ester and triglyceride/protein ratios, which were significantly higher in NIDDM patients than in controls and were not modified by insulin therapy. The persistence of insulin resistance and of high neutral lipid exchanges between triglyceride rich lipoproteins and HDL in insulin-treated NIDDM

patients probably explain the inefficiency of insulin therapy to correct HDL apoA-I metabolic abnormalities.

Publication Types:

- Clinical Trial
- Controlled Clinical Trial

PMID: 10996359 [PubMed - indexed for MEDLINE]

Display Abstract

Show 20

Sort by

Send to

Write to the Help Desk

NCBI | NLM | NIH

Department of Health & Human Services

Privacy Statement | Freedom of Information Act | Disclaimer

May 16 2005 17:16:29